Specification Amendments

Please replace the paragraph beginning on page 42, line 9 with the following paragraph:

B1

Table 2: Confluent monolayers of BSC1 cells ($5X10^5$ cells/well) were infected with moi=1.0 of fowlpox virus strain HP1. Two hours later supernatant was removed, cells were washed 2X with Opti-Mem I media, and transfected using lipofectamine with 600ng vaccinia strain WR genomic DNA either alone, or with 1:1 or 1:10 (vaccinia:plasmid) molar ratios of plasmid pE/Lova. This plasmid contains a fragment of the ovalbumin cDNA, which encodes the SIINFEKL (SEQ ID NO:10) epitope, known to bind with high affinity to the mouse class I MHC molecule K^b. Expression of this minigene is controlled by a strong, synthetic Early/Late vaccinia promoter. This insert is flanked by vaccinia tk DNA. Three days later cells were harvested, and virus extracted by three cycles of freeze/thaw in dry ice isopropanol/ 37°C water bath. Crude virus stocks were titered by plaque assay on human TK- 143B cells with and without BrdU.

Please replace the paragraph beginning on page 52, line 17 with the following paragraph:

B2

Following 12 hours of infection with the recombinant vaccinia virus expressing ovalbumin peptide, ovalbumin peptide-specific CTL, derived by repeated *in vitro* stimulation of ovalbumin primed splenic T cells with the immunodominant ovalbumin SIINFEKL (SEQ ID NO:10) peptide, were added for 30 min.

Please replace the paragraph beginning on page 92, line 15 with the following paragraph:

Use of the thymidine kinase gene as the insertion site for foreign DNA allows implementation of selection protocols for distinguishing recombinants from helper or wild type genomes. The level of tk expression in v7.5/tk and vEL/tk should be much higher than in vaccinia WR or vNot/tk. However, the ApaI site at the beginning of the tk gene in v7.5/tk and vEL/tk was formed from vNot/tk by adding extra nucleotides at the NotI site. The additional nucleotides increase the amino acid sequence at the N terminus of the wild type tk gene from Met-Asn-Gly to Met-Gly-Pro-Ala-Ala-Asn-Gly (SEQ ID NO:38) in v7.5/tk and vEL/tk. Modifications in the expression level and N terminal amino acid sequence of the thymidine kinase gene may increase (more protein) or decrease (different sequence) the sensitivity of the virus to bromodeoxyuridine. Plaques, albeit smaller, were observed with v7.5/tk and vEL/tk infection at a concentration of bromodeoxyuridine sufficient to completely suppress plaque formation for wild type vaccinia WR. Plaque formation was suppressed at five-fold higher concentrations of bromodeoxyuridine, a level of drug that does not interfere with the viability of the cells or impede the ability of tk^- virus to form plaques. The explanation for the altered sensitivity to bromodeoxyuridine awaits further characterization of the protein as the altered thymidine kinase gene may have a different reaction rate for formation of the triphosphate form of the bromodeoxyuridine or a reduced ability to bind bromodeoxyuridine.

B3

Please replace the pending sequence listing with the attached substitute sequence listing at the end of the application.